CLAIMS OF THE INVENTION

That which is claimed is:

1. A pharmaceutical composition for increasing concentrations of chemokines to reduce entry of HIV virus into mononuclear cells through binding of chemokine binding receptors, the composition comprising at least one G1 phase arresting compound in an amount sufficient to increase concentrations of extracellular β-chemokines.

2. The pharmaceutical composition of claim 1, further comprising at least one antiviral agent.

3. The pharmaceutical composition of claim 1, wherein the G1 phase arresting compound is a member selected from the group consisting of sodium butyrate, aphidicolin, hydroxyurea (HU), olomoucine, roscovitine, tocopherols, tocotrienols, and rapamycin (RAPA).

4. The pharmaceutical composition of claim 2, wherein the antiviral agent is an HIV antiviral agent.

5. The pharmaceutical composition of claim 4, wherein the HIV antiviral agent is a nucleoside RT inhibitor, CCR5 inhibitors/antagonist, viral entry inhibitor or functional equivalent thereof.

6. The pharmaceutical composition of claim 2, wherein the antiviral agent is at least one member selected from the group consisting of: Zidovudine (ZDV, AZT), Lamivudine (3TC), Stavudine (d4T), Didanosine (ddI), Zalcitabine (ddC), Abacavir (ABC), Emirivine (FTC), Tenofovir (TDF), Delaviradine (DLV), Efavirenz (EFV), Nevirapine (NVP), Fuzeon (T-20), Saquinavir (SQV), Ritonavir (RTV), Indinavir (IDV), Nelfinavir (NFV), Amprenavir (APV), Lopinavir (LPV), Atazanavir, Combivir (ZDV/3TC), Kaletra (RTV/LPV), Trizivir (ZDV/3TC/ABC), SCH-C, SCH-D, PRO 140, TAK 779, TAK-220,

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RANTES analogs, AK602, UK-427, 857, monoclonal antibodies, NB-2, NB-64, T-649, T-1249, and functional analog thereof.

7. The pharmaceutical composition of claim 4, wherein the compound is administered orally, rectally, nasally, topically, vaginally or parenterally.

8. The pharmaceutical composition of claim 4, wherein the antiviral agent comprises tenofovir in combination with HU.

9. The pharmaceutical composition of claim 4, wherein the antiviral agent comprises tenofovir, 3TC and Efavirenz in combination with HU.

10. The pharmaceutical compositions of claim 2, wherein the composition is administered alone and in combination with the antiviral agent in a cyclic therapy program.

11. A method for inducing increased levels of anti-HIV β-chemokines in activated lymphocytes, the method comprising:

administering a composition comprising at least one G1 phase arresting agent in an effective amount to increase levels of anti-HIV β-chemokines, wherein the increased levels of anti-HIV β-chemokines bind to β-chemokine receptors thereby reducing viral entry of HIV.

12. The method according to claim 11, wherein the G1 phase arresting agent is a member selected from the group consisting of: sodium butyrate, aphidicolin, hydroxyurea (HU), olomoucine, roscovitine, tocopherols, tocotrienols, and rapamycin (RAPA).

13. The method according to claim 11, further comprising at least one antiviral agent.

14. The method according to claim 13, wherein the antiviral agent is an HIV antiviral agent.

15. The method according to claim 14, wherein the HIV antiviral agent is a nucleoside RT inhibitor, CCR5 inhibitors/antagonist, viral entry inhibitor or functional equivalent thereof.

16. The method according to claim 13, wherein the at least one antiviral agent is a member selected from the group consisting of: Zidovudine (ZDV, AZT), Lamivudine (3TC), Stavudine (d4T), Didanosine (ddI), Zalcitabine (ddC), Abacavir (ABC), Emirivine (FTC), Tenofovir (TDF), Delaviradine (DLV), Efavirenz (EFV), Nevirapine (NVP), Fuzeon (T-20), Saquinavir (SQV), Ritonavir (RTV), Indinavir (IDV), Nelfinavir (NFV), Amprenavir (APV), Lopinavir (LPV), Atazanavir, Combivir (ZDV/3TC), Kaletra (RTV/LPV), Trizivir (ZDV/3TC/ABC), SCH-C, SCH-D, PRO 140, TAK 779, TAK-220, RANTES analogs, AK602, UK-427, 857, monoclonal antibodies, NB-2, NB-64, T-649, T-1249, and functional analog thereof.

17. The method according to claim 13, wherein the compound is administered orally, rectally, nasally, topically, vaginally or parenterally.

18. A method for modifying synthesis of a receptor ligand to alter extracellular recognition of a receptor by an infectious agent, the method comprising:

administering to a cell at least one G1 phase arresting agent in an amount sufficient to increase levels of the receptor ligand, thereby inhibiting entry of the infectious agent via the receptor.

19. The method according to claim 18, wherein the receptor ligand comprises a β-chemokine.

20. The method according to claim 19, wherein the chemokine comprises MIP-1α, MIP-1β and RANTES.

21. The method according to claim 18, wherein the infectious agent is HIV.

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22. The method according to claim 18, further comprising administering at least one antiviral agent.

23. The method according to claim 22, wherein G1 phase arresting compound is a member selected from the group consisting of: sodium butyrate, aphidicolin, hydroxyurea (HU), olomoucine, roscovitine, tocopherols, tocotrienols, and rapamycin (RAPA).

24. The method according to claim 22, wherein the antiviral agent is a nucleoside RT inhibitor, CCR5 inhibitors/antagonist, viral entry inhibitor or functional equivalent thereof.

25. The method according to claim 22, wherein the antiviral agent is at least one member selected from the group consisting of: Zidovudine (ZDV, AZT), Lamivudine (3TC), Stavudine (d4T), Didanosine (ddI), Zalcitabine (ddC), Abacavir (ABC), Emirivine (FTC), Tenofovir (TDF), Delaviradine (DLV), Efavirenz (EFV), Nevirapine (NVP), Fuzeon (T-20), Saquinavir (SQV), Ritonavir (RTV), Indinavir (IDV), Nelfinavir (NFV), Amprenavir (APV), Lopinavir (LPV), Atazanavir, Combivir (ZDV/3TC), Kaletra (RTV/LPV), Trizivir (ZDV/3TC/ABC), SCH-C, SCH-D, PRO 140, TAK 779, TAK-220, RANTES analogs, AK602, UK-427, 857, monoclonal antibodies, NB-2, NB-64, T-649, T-1249, and functional analog thereof.

26. The method according to claim 22, wherein the compound is administered orally, rectally, nasally, topically, vaginally or parenterally.

27. A therapeutically effective method of combating a virus infection, the method comprising:

administering to a subject a therapeutically effective amount of a composition comprising a G1 phase arresting compound in a concentration sufficient to induce increased levels and availability of β-chemokines thereby antagonizing the function of a chemokine receptor and reducing replication of the virus infection.

28. The method according to claim 27, wherein the β-chemokines comprise MIP-1α,

MIP-1β or RANTES.

29. The method according to claim 27, wherein the chemokine receptor is CCR5.

30. The method according to claim 27, wherein G1 phase arresting compound is a member selected from the group consisting of: sodium butyrate, aphidicolin, hydroxyurea (HU), olomoucine, roscovitine, tocopherols, tocotrienols, and rapamycin (RAPA).

31. The method according to claim 27, further comprising administering an effective amount of at least one HIV antiviral agent.

32. The method according to claim 31, wherein the antiviral agent is a nucleoside RT inhibitor, CCR5 inhibitors/antagonist, viral entry inhibitor or functional equivalent thereof.

33 The method according to claim 31, wherein the antiviral agent is at least one member selected from the group consisting of: Zidovudine (ZDV, AZT), Lamivudine (3TC), Stavudine (d4T), Didanosine (ddI), Zalcitabine (ddC), Abacavir (ABC), Emirivine (FTC), Tenofovir (TDF), Delaviradine (DLV), Efavirenz (EFV), Nevirapine (NVP), Fuzeon (T-20), Saquinavir (SQV), Ritonavir (RTV), Indinavir (IDV), Nelfinavir (NFV), Amprenavir (APV), Lopinavir (LPV), Atazanavir, Combivir (ZDV/3TC), Kaletra (RTV/LPV), Trizivir (ZDV/3TC/ABC), SCH-C, SCH-D, PRO 140, TAK 779, TAK-220, RANTES analogs, AK602, UK-427, 857, monoclonal antibodies, NB-2, NB-64, T-649, T-1249, and functional analog thereof.

34. The method according to claim 27, further comprising administering an effective amount of an HIV vaccine.

35. The method according to claim 34, wherein the HIV vaccine and the G1 phase arresting agent are administered concurrently.

36. The method according to claim 32, wherein the antiviral agent and the G1 phase

arresting agent are administered concurrently.

37. A method of maintaining viral control of an HIV infection, the method comprising:

administering at least one antiviral agent in combination with at least one G1 phase arresting compound, wherein the G1 phase aresting compound is in a concentration sufficient to increase levels of β-chemokines.

38. The method according to claim 37, wherein the at least one antiviral agent and the at least one G1 phase arresting compound are administered concurrently.

39. The method according to claim 38, wherein the G1 phase arresting compound is a member selected from the group consisting of: sodium butyrate, aphidicolin, hydroxyurea (HU), olomoucine, roscovitine, tocopherols, tocotrienols, and rapamycin (RAPA).

40. The method according to claim 39, wherein the antiviral agent is at least one member selected from the group consisting of: Zidovudine (ZDV, AZT), Lamivudine (3TC), Stavudine (d4T), Didanosine (ddl), Zalcitabine (ddC), Abacavir (ABC), Emirivine (FTC), Tenofovir (TDF), Delaviradine (DLV), Efavirenz (EFV), Nevirapine (NVP), Fuzeon (T-20), Saquinavir (SQV), Ritonavir (RTV), Indinavir (IDV), Nelfinavir (NFV), Amprenavir (APV), Lopinavir (LPV), Atazanavir, Combivir (ZDV/3TC), Kaletra (RTV/LPV), Trizivir (ZDV/3TC/ABC), SCH-C, SCH-D, PRO 140, TAK 779, TAK-220, RANTES analogs, AK602, UK-427, 857, monoclonal antibodies, NB-2, NB-64, T-649, T-1249, and functional analog thereof.

41. The method according to claim 41, wherein the G1 phase arresting agent is HU.

42. The method according to claim 41, wherein the G1 phase arresting agent is rapamycin.

43. A therapeutically effective method to inhibit replication of HIV in a HIV infected subject, the method comprising:

a) administering at least one G1 phase arresting agent in a concentration sufficient to increase concentration of extracellular β-chemokines for a first predetermined time period; and

b) administering the G1 phase agent with at least one antiviral agent, for a second predetermined time period, wherein the first and second time periods are sequential in a cyclic schedule.

44. The therapeutically effective method according to claim 43, wherein the G1 phase arresting agent is a member selected from the group consisting of: sodium butyrate, aphidicolin, hydroxyurea (HU), olomoucine, roscovitine, tocopherols, tocotrienols, and rapamycin (RAPA).

45. The therapeutically effective method according to claim 43, wherein the antiviral agent is a nucleoside RT inhibitor, CCR5 inhibitors/antagonist, viral entry inhibitor or functional equivalent thereof.

46. The therapeutically effective method according to claim 43, wherein the antiviral agent is at least one member selected from the group consisting of: Zidovudine (ZDV, AZT), Lamivudine (3TC), Stavudine (d4T), Didanosine (ddI), Zalcitabine (ddC), Abacavir (ABC), Emirivine (FTC), Tenofovir (TDF), Delaviradine (DLV), Efavirenz (EFV), Nevirapine (NVP), Fuzeon (T-20), Saquinavir (SQV), Ritonavir (RTV), Indinavir (IDV), Nelfinavir (NFV), Amprenavir (APV), Lopinavir (LPV), Atazanavir, Combivir (ZDV/3TC), Kaletra (RTV/LPV), Trizivir (ZDV/3TC/ABC), SCH-C, SCH-D, PRO 140, TAK 779, TAK-220, RANTES analogs, AK602, UK-427, 857, monoclonal antibodies, NB-2, NB-64, T-649, T-1249, and functional analog thereof.

47. The therapeutic method according to claim 43, wherein the cyclic schedule comprises:

a) administering a combination of at least one antiviral agent and at least one G1 phase arresting agent to the HIV infected subject for a predetermined first time period;

b) administering the at least one G1 phase arresting compound to the HIV infected

subject for a second predetermined time period;

c) administering the combination of the antiviral agent and G1 phase arresting agent to the HIV infected subject for a predetermined third time period, which is less than the first period;

d) administering the G1 phase arresting compound to the HIV infected subject for a fourth predetermined time period which is less than the second time period; and

e) maintaining the cyclic schedule of steps c and d until replication of the HIV increases to a predetermined level.

48. A method of preventing HIV infection in a subject potentially exposed to HIV, the method comprising:

administering to the subject at least one G1 phase arresting compound in an effective amount to increase levels of β-chemokines thereby inhibiting binding of HIV to β-chemokine receptors.

49. The method according to claim 48, wherein the G1 phase arresting agent is a member selected from the group consisting of: sodium butyrate, aphidicolin, hydroxyurea (HU), olomoucine, roscovitine, tocopherols, tocotrienols, and rapamycin (RAPA).

50. The method according to claim 48, wherein the G1 phase arresting agent is administered orally, rectally, nasally, topically, vaginally or parenterally.

51. A therapeutically effective method to reduce an effective dosage of an HIV antiviral agent, the method comprising substituting the antiviral agent with a G1 phase arresting compound; augmenting the antiviral agent with a G1 phase arresting compound; or substituting a portion of the antiviral agent with a G1 phase arresting compound, wherein the G1phase arresting compound is in a concentration sufficient to increase concentration of extracellular β-chemokines.

52. The therapeutically effective method according to claim 51, wherein the G1 phase arresting agent is a member selected from the group consisting of: sodium butyrate,

aphidicolin, hydroxyurea (HU), olomoucine, roscovitine, tocopherols, tocotrienols, and rapamycin (RAPA).

53. The therapeutically effective method according to claim 51, wherein the antiviral agent is a nucleoside RT inhibitor, CCR5 inhibitors/antagonist, viral entry inhibitor or functional equivalent thereof.

54. The therapeutically effective method according to claim 51, wherein the antiviral agent is at least one member selected from the group consisting of: Zidovudine (ZDV, AZT), Lamivudine (3TC), Stavudine (d4T), Didanosine (ddI), Zalcitabine (ddC), Abacavir (ABC), Emirivine (FTC), Tenofovir (TDF), Delaviradine (DLV), Efavirenz (EFV), Nevirapine (NVP), Fuzeon (T-20), Saquinavir (SQV), Ritonavir (RTV), Indinavir (IDV), Nelfinavir (NFV), Amprenavir (APV), Lopinavir (LPV), Atazanavir, Combivir (ZDV/3TC), Kaletra (RTV/LPV), Trizivir (ZDV/3TC/ABC), SCH-C, SCH-D, PRO 140, TAK 779, TAK-220, RANTES analogs, AK602, UK-427, 857, monoclonal antibodies, NB-2, NB-64, T-649, T-1249, and functional analog thereof.